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Set	Items	Description
? s	enterotoxin and (outer()	membrane())protein)
	9828	ENTEROTOXIN
	99583	OUTER
	698487	MEMBRANE
	1976373	PROTEIN
	7409	OUTER(W)MEMBRANE(W)PROTEIN
S1	29	ENTEROTOXIN AND (OUTER())MEMBRANE()PROTEIN)
? t	sl/7/1-29	

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0020524051 BIOSIS NO.: 200800570990
Construction of prokaryotic expression vector for LTB-MOMP fusion gene
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JOURNAL: Chinese Journal of Biologicals 21 (6): p449-451, 456 JUN 2008 2008
ISSN: 1004-5503
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Chinese

ABSTRACT: Objective To construct an expression vector for fusion gene of heat-labile enterotoxin (LTB) B subunit (LTB) and major outer membrane protein (MOMP) of Chlamydia psittaci (CP) and express the fusion gene in prokaryotic cells. Methods Amplify LTB and MOMP genes from EMD299 and CP by PCR respectively and link with a flexible peptide (Gly(4)Ser)(3) then insert into vector pET-28a. Identify the constructed recombinant plasmid by restriction analysis and transform to E. coli Rosetta for expression under induction of IPTG. Purify the expressed protein and identify by SDS-PAGE and Western blot. Results The expressed LTB-MOMP fusion protein, with a relative molecular mass of about 54 000, contained 42% of total somatic protein and showed good antigenic specificity. Conclusion The expression vector for LTB-MOMP fusion gene was successfully constructed and expressed in prokaryotic cells.

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0020416563 BIOSIS NO.: 200800463502
Nasal vaccination with the 40-kilodalton outer membrane protein of Porphyromonas gingivalis and a nontoxic chimeric enterotoxin adjuvant induces long-term protective immunity with reduced levels of immunoglobulin E antibodies
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Yoshikazu; Kiyono Hiroshi; Yamamoto Masafumi (Reprint)
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JOURNAL: Infection and Immunity 76 (6): p2777-2784 JUN 2008 2008
ITEM IDENTIFIER: doi:10.1128/IAI.01502-07
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LANGUAGE: English

ABSTRACT: In this study, we demonstrated that the 40-kDa outer membrane protein of *Porphyromonas gingivalis* (40-kDa OMP) nasally administered with a nontoxic chimeric adjuvant that combines the A subunit of mutant cholera toxin E112K with the pentameric B subunit of heat-labile enterotoxin from enterotoxigenic *Escherichia coli* (mCTA/LTB) elicited a long-term protective immune response. Immunization with the 40-kDa OMP and mCTA/LTB induced high levels of 40-kDa-OMP-specific immunoglobulin G (IgG) and IgA antibodies (Abs) in sera and elicited a significant IgA anti-40-kDa OMP Ab response in saliva. These Ab responses were maintained for at least 1 year after the immunization. Although using adjuvant mCTA/LTB gave Ab responses in the saliva comparable to those obtained using native cholera toxin (nCT) as the adjuvant, the levels of total IgE and 40-kDa-OMP-specific IgE Abs as well as interleukin-4 levels induced by the immunization with mCTA/LTB were lower than those induced by the immunization with nCT. Importantly, IgG Abs generated by nasal immunization with the 40-kDa OMP plus mCTA/LTB inhibited the coaggregation and hemagglutinin activities of *P. gingivalis*. Furthermore, the mice given nasal 40-kDa OMP plus mCTA/LTB showed a significant reduction of alveolar bone loss caused by oral infection with *P. gingivalis* even 1 year after the immunization compared to the loss in unimmunized mice. Because mCTA/LTB is nontoxic, nasally administered 40-kDa OMP together with mCTA/LTB should be an effective and safe mucosal vaccine against *P. gingivalis* infection in humans and may be an important tool for the prevention of chronic periodontitis.

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0020190758 BIOSIS NO.: 200800237697
The type III secretion system and cytotoxic enterotoxin modulate quorum sensing in *Aeromonas hydrophila*
AUTHOR: Pillai L (Reprint); Sha J; Fadl A A; Galindo C L; Erova T E; Popov V L; Chopra A K
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JOURNAL: Abstracts of the General Meeting of the American Society for Microbiology 105 p447 2005 2005
CONFERENCE/MEETING: 105th General Meeting of the American Society for Microbiology Atlanta, GA, USA June 05 -09, 2005; 20050605
SPONSOR: Amer Soc Microbiol
ISSN: 1060-2011
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LANGUAGE: English

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0020186488 BIOSIS NO.: 200800233427

Role of outer membrane proteins in diarrhea and identification of toxR of
Vibrio tubiashii

AUTHOR: Jean-Gilles J (Reprint); Kothary M H; Flores N C; Curtis S K; Eribo
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JOURNAL: Abstracts of the General Meeting of the American Society for
Microbiology 106 p181 2006 2006

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0020018264 BIOSIS NO.: 200800065203

Biochemical characterization of the enterotoxigenic *Escherichia coli* LeoA
protein

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JOURNAL: Microbiology (Reading) 153 (Part 11): p3776-3784 NOV 2007 2007

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ISSN: 1350-0872

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RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Enterotoxigenic *Escherichia coli* (ETEC) causes enterotoxin-
included diarrhoea and significant mortality. The molecular mechanisms
underlying how the heat-labile enterotoxin (LT) is secreted during
infection are poorly understood. ETEC produce outer-membrane vesicles
(OMVs) containing LT that are endocytosed into host cells. Although OMV
production and protein content may be a regulated component of ETEC
pathogenesis, how LT loading into OMVs is regulated is unknown. The LeoA
protein plays a role in secreting LT from the bacterial periplasm. To
begin to understand the function of LeoA and its role in ETEC H10407
pathogenesis, a site-directed mutant lacking the putative GTP-binding
domain was constructed. The ability of wild-type and mutant LeoA to
hydrolyse GTP *in vitro* was quantified. This domain was found to be
responsible for GTP binding; it is important to LeoA's function in LT
secretion, and may play a modest role in the formation and protein
content of OMVs. Deletion of *leoA* reduced the abundance of OmpX in

outer-membrane protein preparations and of LT in OMVs. Immunoprecipitation experiments revealed that LeoA interacts directly with OmpA, but that the GTP-binding domain is non-essential for this interaction. Deletion of leoA rendered ETEC H10407 non-motile, through apparent periplasmic accumulation of FlhC.

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0019752946 BIOSIS NO.: 200700412687

Global transcriptional responses of wild-type *Aeromonas hydrophila* and its virulence-deficient mutant in a murine model of infection

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LANGUAGE: English

ABSTRACT: We previously generated a double knockout mutant (act/aopB) of a diarrheal isolate SSU of *A. hydrophila*, in which the genes encoding *Aeromonas* outer-membrane protein B (AopB), a structural component of the type III secretion system (T3SS), and a type II (T2)-secreted cytotoxic enterotoxin gene (act) were deleted. This mutant exhibited minimal virulence in mice, compared to animals infected with wild-type (WT) *A. hydrophila*. Based on microarray analyses, WT *A. hydrophila* altered the expression of 434 and 80 genes in murine macrophages (RAW 264.7) and human colonic epithelial cells (HT-29), respectively. Approximately half of these gene expression alterations were abrogated when host cells were infected instead with the act/aopB mutant. In this study, we used microarrays to examine early host transcriptional responses in spleens of mice infected for 3 h with WT *A. hydrophila* or its act/aopB mutant. Our data indicated that expression of 221 genes was altered (158 up-regulated and 63 down-regulated) in spleens of WT bacteria-infected animals. There were 21 genes that were consistently more highly expressed in WT *A. hydrophila*-infected mice, compared to mice infected with its act/aopB mutant. Ten of these genes were either induced to a lesser extent (e.g., interleukin-6, macrophage inflammatory protein-2, and cyclooxygenase-2), not altered at all (e.g., killer cell lectin-like receptor subfamily B member A), or down-regulated (e.g., cytochrome P450) in animals infected with *A. hydrophila*, compared to phosphate-buffered saline-infected control animals, when the mutant was used instead of the WT. We verified the microarray results at the transcript level by performing real-time reverse transcriptase-polymerase chain reaction on selected genes and at the protein level by enzyme-linked immunosorbent assay (ELISA) and immunohistochemistry. This is the first study demonstrating in vivo gene regulation in mice infected with *A. hydrophila* and the contribution of virulence factors and host responses to the disease process. (C) 2007 Elsevier Ltd. All rights reserved.

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19203771 BIOSIS NO.: 200600549166

Secretory production of therapeutic proteins in Escherichia coli

BOOK TITLE: Methods in Molecular Biology

AUTHOR: Lee Sang Yup (Reprint); Choi Jong Hyun; Lee Sang Jun

BOOK AUTHOR/EDITOR: Smales CM (Editor); James DC (Editor)

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19145389 BIOSIS NO.: 200600490784

Alterations in the virulence potential of enteric pathogens and
bacterial-host cell interactions under simulated microgravity conditions

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JOURNAL: Journal of Toxicology and Environmental Health Part A 69 (14): p
1345-1370 JUL 15 2006 2006

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ABSTRACT: Host immune mechanisms were proposed to decline under microgravity conditions during spaceflights, which might result in severe infections in astronauts. Therefore, it was important to investigate the effects of microgravity on infecting organisms and their interaction with host cells. Data showed that simulated microgravity (SMG) conditions markedly increased production of the enterotoxigenic Escherichia coli (ETEC) heat-labile enterotoxin, which induced fluid secretory responses in a mouse model. SMG also enhanced production of tumor necrosis factor- α in murine macrophages infected with enteropathogenic E. coli (EPEC). In a similar fashion, simulated microgravity conditions augmented the invasive potential of Salmonella enterica serovar typhimurium and enhanced production of tumor necrosis-factor α in S. typhimurium-infected epithelial cells. Furthermore, coculturing of macrophages and S. typhimurium in a simulated microgravity environment resulted in activation of stress-associated mitogen-activated protein kinase kinase 4. Using the antiorthostatic tail

suspension mouse model, which simulates some aspects of microgravity, oral inoculation of *S. typhimurium* markedly reduced the 50% lethal dose compared to mice infected under normal gravitational conditions. Microarray analysis revealed simulated microgravity-induced alterations in the expression of 22 genes in *S. typhimurium*, and protein expression profiles were altered in both EPEC and *S. typhimurium*, based on two-dimensional gel electrophoresis. These studies indicated alterations in the virulence potential of bacteria and in host responses to these pathogens under simulated microgravity conditions, which may represent an important environmental signal. Such studies are essential for better understanding bacterial-host cell interactions, particularly in the context of spaceflights and space habitations of long duration.

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19015642 BIOSIS NO.: 200600361037

Deletion of the genes encoding the type III secretion system and cytotoxic *enterotoxin* alters host responses to *Aeromonas hydrophila* infection

AUTHOR: Fadl Amin A; Galindo Cristi L; Sha Jian; Erova Tatiana E; Houston Clifford W; Olano Juan P; Chopra Ashok K (Reprint)

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RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: In our previous study, we deleted the gene encoding *Aeromonas* *outer membrane protein B* (AopB), a structural component of the type III secretion system (T3SS) from a cytotoxic *enterotoxin* gene (act)-minus diarrheal isolate SSU of *Aeromonas hydrophila*. Our laboratory also molecularly characterized the cytotoxic *enterotoxin* (Act), which is secreted by the bacterium utilizing the type III secretion system (T2SS). The act/aopB mutant exhibited significantly reduced cytotoxicity to cultured cells (e.g. RAW 264.7 murine macrophages and HT-29 human colonic epithelial cells) and was avirulent in mice. In this study, we developed additional *A. hydrophila* mutants in which T3SS-associated ascV and acrV genes were deleted, either individually or in combination with that of the act gene, to examine host-pathogen interactions. A significant reduction in the induction of inflammatory cytokines and chemokines was noted in the sera of mice infected with these mutants when compared to animals infected with wild-type (WT) *A. hydrophila*. After infection with the WT and act/aopB mutant, we performed microarray analyses on RNA from the above-mentioned murine macrophages and human colonic epithelial cells to examine global cellular transcriptional responses. Based on three independent experiments, WT *A. hydrophila* altered the expression of 434 genes in RAW 264.7 cells and 80 genes in HT-29 cells. Alteration in the expression of 209 macrophage and 32 epithelial cell genes was reduced when the act/aopB mutant was used, compared to when cells were infected with the WT bacterium, indicating the involvement of Act and/or AopB in transcriptional regulation of these genes. We verified up-regulation of

15 genes by real-time reverse transcriptase-polymerase chain reaction and confirmed *A. hydrophila* WT-versus mutant-induced production of cytokines/chemokines in supernatants from RAW 264.7 and HT-29 cells. This is the first description of host cell transcriptional responses to *A. hydrophila* infection. (c) 2006 Elsevier Ltd. All rights reserved.

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18645574 BIOSIS NO.: 200510340074
The type III secretion system and cytotoxic enterotoxin alter the virulence of *Aeromonas hydrophila*
AUTHOR: Sha Jian; Pillai Lakshmi; Fadl Amin A; Galindo Cristi L; Erova Tatiana E; Chopra Ashok K (Reprint)
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JOURNAL: Infection and Immunity 73 (10): p6446-6457 OCT 2005 2005
ISSN: 0019-9567
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LANGUAGE: English

ABSTRACT: Many gram-negative bacteria use a type III secretion system (TTSS) to deliver effector proteins into host cells. Here we report the characterization of a TTSS chromosomal operon from the diarrheal isolate SSU of *Aeromonas hydrophila*. We deleted the gene encoding *Aeromonas* outer membrane protein B (AopB), which is predicted to be involved in the formation of the TTSS translocon, from wild-type (WT) *A. hydrophila* as well as from a previously characterized cytotoxic enterotoxin gene (act) -minus strain of *A. hydrophila*, thus generating aopB and act/aopB isogenic mutants. The act gene encodes a type II-secreted cytotoxic enterotoxin (Act) that has hemolytic, cytotoxic, and enterotoxic activities and induces lethality in a mouse model. These isogenic mutants (aopB, act, and act/aopB) were highly attenuated in their ability to induce cytotoxicity in RAW 264.7 murine macrophages and HT-29 human colonic epithelial cells. The act/aopB mutant demonstrated the greatest reduction in cytotoxicity to cultured cells after 4 h of infection, as measured by the release of lactate dehydrogenase enzyme, and was avirulent in mice, with a 90% survival rate compared to that of animals infected with Act and AopB mutants, which caused 50 to 60% of the animals to die at a dose of three 50% lethal doses. In contrast, WT *A. hydrophila* killed 100% of the mice within 48 h. The effects of these mutations on cytotoxicity could be complemented with the native genes. Our studies further revealed that the production of lactones, which are involved in quorum sensing (QS), was decreased in the act (32%) and aopB (64%) mutants and was minimal (only 8%) in the act/aopB mutant, compared to that of WT *A. hydrophila* SSU. The effects of act and aopB gene deletions on lactone production could also be complemented with the native genes, indicating specific effects of Act and the TTSS on lactone production. Although recent studies with other bacteria have indicated TTSS regulation by QS, this is the first report describing a correlation between the TTSS and Act of *A. hydrophila* and the production of lactones.

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18049165 BIOSIS NO.: 200400419954
Construction and high expression of an act-OmpTS fusion vector of *Aeromonas hydrophila*
AUTHOR: He Ming-Xiao (Reprint); Ye Qiao-Zhen; Chen Cheng; Xie Jun-Feng; He Jian-guo
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JOURNAL: Acta Hydrobiologica Sinica 28 (2): p169-173 March 2004 2004
MEDIUM: print
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LANGUAGE: Chinese

ABSTRACT: Cytotoxic **enterotoxin** and **outer** **membrane** **protein** have been proved to be protective antigens against *Aeromonas hydrophila*. Primers P1, P2, P3 and P4 were designed based on the sequences of cytotoxic **enterotoxin** gene (act) and outer membrane gene (OmpTS) of *Aeromonas hydrophila* in Genbank (primers P1, P2 for act, primers P3, P4 for ompTS). The genomic DNA of a strain of *Aeromonas hydrophila* isolated in Guangdong Province was extracted and used as PCR template. Then the partial act fragment and ompTS fragment without the signal sequence were amplified separately and purified by DNA agarose gel purification Kit. With these two fragments mixed together as the template, one target fragment about 2.1 kb was amplified with primer P1 and P4 after the second step PCR amplification. A linker, (Gly4Ser)3, was inserted between these two genes. The down stream primer of act overlapped the upstream primer of OmpTS in 21 bp and the linker (Gly4Ser)3 was encoded by this two primers together. The 2.1 kb fragment was digested by BamH I and Hind III, ligated into BamH I /Hind III linearized pQE-30 plasmid (Qiagen Co.). pQE30/act-GS-OmpTS, an expression vector with the fusion fragment was then constructed. After transformed into *E. coli* M15 (pREP4) and induced with IPTG, pQE30/act-GS-OmpTS was hyper-expressed. To optimize the expression of the recombinant fusion protein, expression conditions ranging in salinity, pH value, IPTG concentration, induced time, induced temperature and medium type were tested. The optimal condition was proved to be pH7, 0.02% NaCl, LB, 30°C and 0.2 mmol/L IPTG for 3-5h. The recombinant fusion protein (Act-GS-OmpTS) exhibited a molecular weight of about 81.0 kDa in 12% SDS-PAGE, which was identical to what had been anticipated. Scanned by CS-950 spot scanning densitometer (SHIMADZU(TM)), the band of the recombinant fusion protein showed about 42% of total *E. coli* proteins. Western blot analysis shown that rabbit polyclonal antibody to Act and OmpTS all reacted with Act-GS-OmpTS, which indicated the fusion protein may have similar epitopes to those of the natural proteins.

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17698735 BIOSIS NO.: 200400079492

Characterization of *Vibrio fluvialis*-like strains implicated in limp lobster disease.

AUTHOR: Tall B D (Reprint); Fall S; Pereira M R; Ramos-Valle M; Curtis S K; Kothary M H; Chu D M T; Monday S R; Kornegay L; Donkar T; Prince D; Thunberg R L; Shangraw K A; Hanes D E; Khambaty F M; Lampel K A; Bier J W; Bayer R C

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December 2003 2003

MEDIUM: print

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RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Studies were undertaken to characterize and determine the pathogenic mechanisms involved in a newly described systemic disease in *Homarus americanus* (American lobster) caused by a *Vibrio fluvialis*-like microorganism. Nineteen isolates were obtained from eight of nine lobsters sampled. Biochemically, the isolates resembled *V. fluvialis*, and the isolates grew optimally at 20degreeC; none could grow at temperatures above 23degreeC. The type strain (LAMA) displayed a thermal reduction time (D value) of 5.77 min at 37degreeC. All of the isolates required at least 1% NaCl for growth. Collectively, the data suggest that these isolates may embody a new biotype. Pulsed-field gel electrophoresis (PFGE) analysis of the isolates revealed five closely related subgroups. Some isolates produced a sheep hemagglutinin that was neither an outer membrane protein nor a metalloprotease. Several isolates possessed capsules. The isolates were highly susceptible to a variety of antibiotics tested. However, six isolates were resistant to erythromycin. Seventeen isolates harbored plasmids. Lobster challenge studies revealed that the 50% lethal dose of a plasmid-positive strain was 100-fold lower than that of a plasmid-negative strain, suggesting that the plasmid may enhance the pathogenicity of these microorganisms in lobsters. Microorganisms that were recovered from experimentally infected lobsters exhibited biochemical and PFGE profiles that were indistinguishable from those of the challenge strain. Tissue affinity studies demonstrated that the challenge microorganisms accumulated in heart and midgut tissues as well as in the hemolymph. Culture supernatants and polymyxin B lysates of the strains caused elongation of CHO cells in tissue culture, suggesting the presence of a hitherto unknown enterotoxin. Both plasmid-positive and plasmid-negative strains caused significant dose-related intestinal fluid accumulations in suckling mice. Absence of viable organisms in the intestinal contents of mice suggests that these microorganisms cause diarrhea in mice by intoxication rather than by an infectious process. Further, these results support the thermal reduction data at 37degreeC and suggest that the mechanism(s) that led to fluid accumulation in mice differs from the disease process observed in lobsters by requiring neither the persistence of viable microorganisms nor the presence of plasmids. In summary, results of lobster studies satisfy Koch's postulates at the organismal and molecular levels; the findings support the hypothesis that these *V.*

fluvialis-like organisms were responsible for the originally described systemic disease, which is now called limp lobster disease.

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15979740 BIOSIS NO.: 200100151579
Molecular analysis of *Vibrio cholerae* O1, O139, non-O1, and non-O139 strains: Clonal relationships between clinical and environmental isolates
AUTHOR: Singh D V (Reprint); Matte Maria H; Matte G R; Jiang Sunny; Sabeena F; Shukla B N; Sanyal S C; Huq A; Colwell R R
AUTHOR ADDRESS: Rajiv Gandhi Centre for Biotechnology, Jagathy, Thiruvananthapuram, KER, 695 014, India**India
JOURNAL: Applied and Environmental Microbiology 67 (2): p910-921 February, 2001 2001
MEDIUM: print
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DOCUMENT TYPE: Article
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LANGUAGE: English

ABSTRACT: A total of 26 strains of *Vibrio cholerae*, including members of the O1, O139, and non-O1, non-O139 serogroups from both clinical and environmental sources, were examined for the presence of genes encoding cholera toxin (ctxA), zonula occludens toxin (zot), accessory cholera enterotoxin (ace), hemolysin (hlyA), NAG-specific heat-stable toxin (st), toxin-coregulated pilus (tcpA), and outer membrane protein (ompU), for genomic organization, and for the presence of the regulatory protein genes tcpI and toxR in order to determine relationships between epidemic serotypes and sources of isolation. While 22 of the 26 strains were hemolytic on 5% sheep blood nutrient agar, all strains were PCR positive for hlyA, the hemolysin gene. When multiplex PCR was used, all serogroup O1 and O139 strains were positive for tcpA, ompU, and tcpI. All O1 and O139 strains except one O1 strain and one O139 strain were positive for the ctxA, zot, and ace genes. Also, O1 strain VO3 was negative for the zot gene. All of the non-O1, non-O139 strains were negative for the ctxA, zot, ace, tcpA, and tcpI genes, and all of the non-O1, non-O139 strains except strain VO26 were negative for ompU. All of the strains except non-O1, non-O139 strain VO22 were PCR positive for the gene encoding the central regulatory protein, toxR. All *V. cholerae* strains were negative for the NAG-specific st gene. Of the nine non-ctx-producing strains of *V. cholerae*, only one, non-O1, non-O139 strain VO24, caused fluid accumulation in the rabbit ileal loop assay. The other eight strains, including an O1 strain, an O139 strain, and six non-O1, non-O139 strains, regardless of the source of isolation, caused fluid accumulation after two to five serial passages through the rabbit gut. Culture filtrates of all non-cholera-toxigenic strains grown in AKI media also caused fluid accumulation, suggesting that a new toxin was produced in AKI medium by these strains. Studies of clonality performed by using enterobacterial repetitive intergenic consensus sequence PCR, Box element PCR, amplified fragment length polymorphism (AFLP), and pulsed-field gel electrophoresis (PFGE) collectively indicated that the *V. cholerae* O1 and O139 strains had a clonal origin, whereas the non-O1, non-O139 strains belonged to different clones. The clinical isolates closely resembled environmental isolates in their genomic patterns.

Overall, there was an excellent correlation among the results of the PCP, AFLP, and PFGE analyses, and individual strains derived from clinical and environmental sources produced similar fingerprint patterns. From the results of this study, we concluded that the non-cholera-toxin-producing strains of *V. cholerae*, whether of clinical or environmental origin, possess the ability to produce a new secretogenic toxin that is entirely different from the toxin produced by toxigenic *V. cholerae* 01 and 0139 strains. We also concluded that the aquatic environment is a reservoir for *V. cholerae* 01, 0139, non-01, and non-0139 serogroup strains.

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15559001 BIOSIS NO.: 200000277314

Mutation of aromatic amino acid residues located at the amino- and carboxy-termini of *Escherichia coli* heat-stable enterotoxin Ip reduces the efficiency of the toxin to cross the outer membrane

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JOURNAL: Microbiology and Immunology 44 (6): p481-488 2000 2000

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RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Heat-stable enterotoxin Ip (STIp) of *Escherichia coli* is synthesized as a precursor form consisting of pre- (amino acid residues 1 to 19), pro- (amino acid residues 20 to 54) and mature (amino acid residues 55 to 72) regions. Mature STIp (bioactive STIp) is formed in the periplasmic space after the precursor is proteolytically processed and the mature STIp translocates across the outer membrane through the secretory system including TolC, an outer membrane protein of *E. coli*. However, it remains unknown how the mature STIp is recognized by this secretory system. In this study, we investigated the amino acid residues of STIp involved in its translocation across the outer membrane. We prepared mutant STIp genes by site-directed mutagenesis and analyzed translocation of the mutant STIps across the outer membrane. Deletion of the Phe or Tyr residue at position 3 or 18, respectively, decreased the efficiency of translocation of STIp across the outer membrane. To confirm the involvement of these amino acid residues, we further mutated the codons for these amino acid residues to that for Gly. These mutations also decreased the efficiency of extracellular secretion of STIp. In contrast, substitution of Phe-3 and Tyr-18 with Tyr and Phe, respectively, did not affect the efficiency of translocation of the toxin. These results indicated that the aromatic amino acid residues at positions 3 and 18 in the mature region are important for the ability of STIp to cross the outer membrane.

1/7/15

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14718121 BIOSIS NO.: 199800512368

Need for TolC, an Escherichia coli outer membrane protein, in the secretion of heat-stable enterotoxin I across the outer membrane

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JOURNAL: Microbial Pathogenesis 25 (3): p111-120 Sept., 1998 1998

MEDIUM: print

ISSN: 0882-4010

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RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Escherichia coli heat-stable enterotoxin (STlp) is a typical extracellular toxin consisting of 18 amino acid residues synthesized as a precursor of pre (amino acid residues 1 to 19), pro (amino acid residues 20 to 54), and mature (amino acid residues 55 to 72) regions. STlp synthesized in the cytoplasm must cross the inner and outer membranes to migrate into the extracellular environment. Previous studies showed that the precursor translocates across the inner membrane utilizing the general export pathway consisting of Sec proteins. However, it remains unclear how it crosses the outer membrane. In this study, we examined the effects of mutation of the tolC gene which encodes an E. coli outer membrane protein, TolC, on the release of STlp into the extracellular environment. The mutation reduced the amount of STlp released into culture supernatant and increased the amount of STlp accumulated in the periplasm. This indicates that TolC mediates the translocation of STlp across the outer membrane. The inability to transfer STlp in the periplasm into the culture supernatant was restored by introduction of the tolC gene into the mutant cells. In the mouse intestinal loop assay, living cells of the mutants did not show a positive response, but wild-type cells did. These results showed that TolC is involved in the translocation of STlp across the outer membrane.

1/7/16

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14660431 BIOSIS NO.: 199800454678

Epidemiology and properties of heat-stable enterotoxin-producing Escherichia coli serotype O169:H41

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JOURNAL: Epidemiology and Infection 121 (1): p31-42 Aug., 1998 1998

MEDIUM: print

ISSN: 0950-2688

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Enterotoxigenic Escherichia coli (ETEC) serotype O169:H41

organisms have become the most prevalent ETEC in Japan since the first outbreak in 1991. It was assumed that the outbreaks were due to clonal spread of this new ETEC serotype. The relationship of 32 strains isolated from 6 outbreaks were examined for biotype, antibiotic susceptibility, enterotoxigenicity, protein banding pattern, lipopolysaccharide banding pattern, plasmid analysis, and ribotyping. Further, the strains were examined by hemagglutination, surface hydrophobicity, and the ability to adhere to HEp-2 cells. The present study suggests that the outbreaks were caused by multiple clones of STp-producing O169:H41 since they showed differences in ribotype and %outer% %membrane% %protein% banding patterns. The strains did not agglutinate human or bovine red blood cells in a mannose-resistant manner. They adhered to HEp-2 cells in a manner resembling enteroaggregative E. coli. Five strains were examined by dot-blot tests for the colonization factor antigens CFA/I, CS1, CS2, CS3, CS4, CS5, CS6, CS7, PCFO159, PCFO166 and CFA/III. Although four strains expressed CS6, no structure for CS6 was identified. A strain that the anti-CS6 MAbs did not react with could adhere to HEp-2 cells in mannose resistant manner; thus, it is unlikely that CS6 play an important role in the adhesion to the cells. Electron microscopy studies of the O169:H41 strains suggested that curly fimbriae, a possible new colonization factor, may be playing an important role in the adhesion of the bacteria to HEp-2 cells. In conclusion, outbreaks due to ETEC O169:H41 were caused by multiple clones, and the strains should be examined in detail for a possible new colonization factor.

1/7/17

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14211147 BIOSIS NO.: 199800005394

General secretion pathway (eps) genes required for toxin secretion and outer membrane biogenesis in *Vibrio cholerae*

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JOURNAL: Journal of Bacteriology 179 (22): p6994-7003 Nov., 1997 1997

MEDIUM: print

ISSN: 0021-9193

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The general secretion pathway (GSP) of *Vibrio cholerae* is required for secretion of proteins including chitinase, %enterotoxin%, and protease through the outer membrane. In this study, we report the cloning and sequencing of a DNA fragment from *V. cholerae*, containing 12 open reading frames, epsC to -N, which are similar to GSP genes of *Aeromonas*, *Erwinia*, *Klebsiella*, *Pseudomonas*, and *Xanthomonas* spp. In addition to the two previously described genes, epsE and epsM (M. Sandkvist, V. Morales, and M. Bagdasarian, Gene 123: 81-86, 1993; L. J. Overbye, M. Sandkvist, and M. Bagdasarian, Gene 132:101-106, 1993), it is shown here that epsC, epsF, epsG, and epsL also encode proteins essential for GSP function. Mutations in the eps genes result in aberrant %outer% %membrane% %protein% profiles, which indicates that

the GSP, or at least some of its components, is required not only for secretion of soluble proteins but also for proper outer membrane assembly. Several of the Eps proteins have been identified by use of the T7 polymerase-promoter system in *Escherichia coli*. One of them, a pilin-like protein, EpsG, was analyzed also in *V. cholerae* and found to migrate as two bands on polyacrylamide gels, suggesting that in this organism it might be processed or otherwise modified by a prepilin peptidase. We believe that TcpJ prepilin peptidase, which processes the subunit of the toxin-coregulated pilus, TcpA, is not involved in this event. This is supported by the observations that apparent processing of EpsG occurs in a tcpJ mutant of *V. cholerae* and that, when coexpressed in *E. coli*, TcpJ cannot process EpsG although the PilD peptidase from *Neisseria gonorrhoeae* can.

1/7/18

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13775134 BIOSIS NO.: 199799409194

Comparison of the nucleotide sequence of enteroaggregative *Escherichia coli* heat-stable enterotoxin 1 genes among diarrhea-associated *Escherichia coli*

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JOURNAL: FEMS Microbiology Letters 147 (1): p89-95 1997 1997

ISSN: 0378-1097

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The presence of the enteroaggregative *Escherichia coli* (EAggEC) heat-stable enterotoxin 1 (EAST1) gene was investigated in 15 strains each of EAggEC, enteropathogenic *E. coli* (EPEC), EPEC-related strains of non-EPEC serotypes, diffusely adhering *E. coli* (type 1 DAEC) that carries F1845 adhesive pili (or a related adhesin), and enteroinvasive *E. coli* (EIEC) by PCR and colony hybridization. The EAST1 gene or its homologue was present in 53.3% of EAggEC, 20% of EPEC, 13.3% of the EPEC-related strains, and 6.7% of type 1 DAEC. EIEC and *E. coli* unrelated with diarrhea had no gene with sequence similarity to the EAST1 gene. Comparison of the EAST1 gene sequences analyzed in this study as well as those reported previously showed that EAggEC (including strain O42, which was shown to be pathogenic in volunteer experiments), EPEC, type 1 DAEC, type 2 DAEC (which carries the 57-kDa outer membrane protein as an adhesin), and enterotoxigenic *E. coli* shared a common sequence. A variant type of the EAST1 gene sequence was present in the EAggEC strain 17-2 (initially characterized for the EAST1 gene) and in an EPEC-related strain of a non-EPEC serotype. These data suggest that the EAST1 gene or its variant is a virulence gene widely distributed among diarrhea-associated *E. coli*.

1/7/19

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13626677 BIOSIS NO.: 199699260737

Immunogenicity and efficacy against lethal aerosol staphylococcal
enterotoxin B challenge in monkeys by intramuscular and respiratory
delivery of proteosome-toxoid vaccines

AUTHOR: Lowell George H (Reprint); Colleton Curtis; Frost Denzil; Kaminski
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JOURNAL: Infection and Immunity 64 (11): p4686-4693 1996 1996

ISSN: 0019-9567

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Staphylococcal enterotoxin B (SEB), a primary cause of food poisoning, is also a superantigen that can cause toxic shock after traumatic or surgical staphylococcal wound infections or viral influenza-associated staphylococcal superinfections or when aerosolized for use as a potential biologic warfare threat agent. Intranasal or intramuscular (i.m.) immunization with formalinized SEB toxoid formulated with meningococcal outer membrane protein proteosomes has previously been shown to be immunogenic and protective against lethal respiratory or parenteral SEB challenge in murine models of SEB intoxication. Here, it is demonstrated that immunization of nonhuman primates with the proteosome-SEB toxoid vaccine is safe, immunogenic, and protective against lethal aerosol challenge with 15 50% lethal doses of SEB. Monkeys (10 per group) were primed i.m. and given booster injections by either the i.m. or intratracheal route without adverse side effects. Anamnestic anti-SEB serum immunoglobulin G (IgG) responses were elicited in all monkeys, but strong IgA responses in sera and bronchial secretions were elicited both pre- and post-SEB challenge only in monkeys given booster injections intratracheally. The proteosome-SEB toxoid vaccine was efficacious by both routes in protecting 100% of monkeys against severe symptomatology and death from aerosolized-SEB intoxication. These data confirm the safety, immunogenicity, and efficacy in monkeys of parenteral and respiratory vaccination with the proteosome-SEB toxoid, thereby supporting clinical trials of this vaccine in humans. The safety and enhancement of both bronchial and systemic IgA and IgG responses by the proteosome vaccine delivered by a respiratory route are also encouraging for the development of mucosally delivered proteosome vaccines to protect against SEB and other toxic or infectious respiratory pathogens.

1/7/20

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13552439 BIOSIS NO.: 199699186499

Characterization of a novel hemagglutinin of diarrhea-associated
Escherichia coli that has characteristics of diffusely adhering E. coli
and enteroaggregative E. coli

AUTHOR: Yamamoto Tatsuo (Reprint); Wakisaka Noriko; Nakae Taiji; Kamano
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JOURNAL: Infection and Immunity 64 (9): p3694-3702 1996 1996

ISSN: 0019-9567

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: *Escherichia coli* 73-1 (serotype O73:H33) and 5-2 (serotype O89:H-) isolated from patients with diarrhea adhered to tissue culture cells (HeLa and HEP-2) as well as coverslips (plastic and glass) in a diffuse pattern. Adherence of strain 73-1 was mediated by a 110-kbp plasmid designated pEDa1 and correlated with D-mannose-resistant hemagglutinin (MRHA) detected with bovine, sheep, or human erythrocytes. The MRHA region was duplicated on pEDa1 and mediated the production of the 57-kDa outer membrane protein whose N-terminal amino acid sequence was hydrophobic. In accordance with MRHA and adherence, the 57-kDa outer membrane protein was observed best at 37 degree C and to a lesser extent at 25 degree C. In human intestine, adherence to mucus and colonic epithelium was obvious. No detectable pili were observed. The enteroaggregative *E. coli* heat-stable enterotoxin 1 (EAST1) gene, whose nucleotide sequence was 99.1% homologous to that of enteroaggregative *E. coli*, was present adjacent to the MRHA region on pEDa1. Strain 5-2 also exhibited MRHA activities and adherence and had sequences corresponding to those of the MRHA region and EAST1 gene. The data suggest that strain 73-1 (and strain 5-2), which has characteristics of both diffusely adhering *E. coli* and enteroaggregative *E. coli*, possesses a novel hemagglutinin associated with diffuse adherence.

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13502512 BIOSIS NO.: 199699136572

Molecular determinants of *Yersinia* pathogenesis

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JOURNAL: Microbiologia (Madrid) 12 (2): p267-280 1996 1996

ISSN: 0213-4101

DOCUMENT TYPE: Article; Literature Review

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The genus *Yersinia* contains three pathogenic species: *Yersinia pestis*, *Y. pseudotuberculosis* and *Y. enterocolitica*. Even though the three species use different routes to infect their host and provoke diseases of different intensity, they share a common tropism for the lymphoid tissue and they are able to resist the primary immune response of the host. The main genetic determinants involved in this resistance are encoded by a highly conserved 70-kb virulence plasmid. The genes harbored by the pYV plasmid encode the lipoprotein YlpA, the outer membrane protein YadA, and a group of at least 11 secreted proteins called Yops. The pYV plasmid also encodes the apparatus necessary for the secretion of the Yop proteins, as well as those involved in the regulation of Yop synthesis. The Yop proteins are secreted by a specific secretion system which is considered as the

archetype of a new secretion pathway called type III. After their secretion they are immediately internalized into the cytosol of a target eukaryotic cell, which represents a new phenomenon in microbial pathogenesis. The chromosome of *Y. enterocolitica* completes the virulence panoply of the bacteria by encoding an enterotoxin called Yst, fibrillae named Myf and an invasins called Inv.

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11936672 BIOSIS NO.: 199396101088

The Myf fibrillae of *Yersinia enterocolitica*

AUTHOR: Iriarte Maite; Vanooteghem Jean-Claude; Delor Isabelle; Diaz Ramon;

Knutton Stuart; Cornelis Guy R (Reprint)

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JOURNAL: Molecular Microbiology 9 (3): p507-520 1993

ISSN: 0950-382X

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The Myf antigen produced by *Yersinia enterocolitica* appeared as a proteic polymer composed of 21 kDa subunits. By transposon mutagenesis we isolated Myf-defective mutants. Those allowed us to clone and sequence a 4.4 kb chromosomal locus involved in Myf production. This region was found to contain three genes that we called myfA, myfB and myfC. Genes myfB and myfC encode an assembly machine related to those involved in the synthesis of many fimbriae: MyfB, the putative chaperone, possesses the consensus residues of the PapD family and myfC encodes a putative outer-membrane protein. MyfA, the major subunit, was found to be 44% identical to the pH 6 antigen of *Y. pestis*. Myf is thus the *Y. enterocolitica* counterpart of this antigen, but it is by far not so well conserved as the other virulence determinants such as the Yops, suggesting that Myf and pH 6 antigen do not necessarily play the same role in *Y. enterocolitica* and *Y. pestis*. The study of the prevalence of myfA in various species of *Yersinia* revealed that, like the yst enterotoxin gene, its presence is restricted to the pathogenic serotypes of *Y. enterocolitica*. By immunogold labelling, Myf appeared as a layer of extracellular material extending locally 2 μ m from the bacterial surface, indicative of a fibrillar structure.

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11853530 BIOSIS NO.: 199396017946

Comparison of *Vibrio cholerae* serotype O1 strains isolated from patients and the aquatic environment

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JOURNAL: Journal of Tropical Medicine and Hygiene 96 (2): p86-92 1993

ISSN: 0022-5304

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: *Vibrio cholerae* 01 strains of El Tor and Classical biotypes and Ogawa and Inaba serotypes were isolated from both patients and pond water, and latter used by the patients from whom the *V. cholerae* 01 strains had been isolated. Paired strains, i.e. from the patient and from the pond used by the patient, were compared. All strains were found to be non-hydrophobic and agglutinating in ammonium sulphate (2.0-2.5 M). They demonstrated similar antibiogram patterns and plasmids were not detected. Except for one clinical and one environmental strain, all strains caused fluid accumulation in the rabbit ileal loop (RIL). The outer membrane protein profiles of both clinical and environmental strains were nearly identical, except for the presence of an additional 22 kDa polypeptide, observed only in environmental strains. The protein profiles of two environmental isolates, analysed after passage through rabbits by oral feeding, were altered, demonstrating a significant decrease in the number of protein bands after animal passage but with the major protein band pattern remaining unchanged. Each passage strain, however, demonstrated properties similar to the non-passaged culture in cell surface hydrophobicity, plasmid profile, antibiogram patterns, and enterotoxin production.

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11641310 BIOSIS NO.: 199345072292

Role of the transcription activator VirF and the histone-like protein YmoA in the thermoregulation of virulence functions in *Yersinia*

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JOURNAL: Zentralblatt fuer Bakteriologie 278 (2-3): p149-164 1993

CONFERENCE/MEETING: Workshop on Molecular Pathogenesis of Bacteria: Basic

and Applied Aspects Schierke, Germany April 21-24, 1992; 19920421

ISSN: 0934-8840

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RECORD TYPE: Citation

LANGUAGE: English

1/7/25

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10638569 BIOSIS NO.: 199191021460

PLASMIDS AND FACTORS ASSOCIATED WITH VIRULENCE IN ENVIRONMENTAL ISOLATES OF *VIBRIO-CHOLERA* NON-01 IN BANGLADESH

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JOURNAL: Journal of Medical Microbiology 33 (2): p107-114 1990

ISSN: 0022-2615

DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: Plasmid profiles and factors associated with toxigenicity in 51 strains of *Vibrio cholerae* non-O1 isolated from water samples collected in Bangladesh were analyzed. Eleven (21.5%) strains were found to harbour at least one plasmid of 1.7-115 Mda; seven of these strains shared a 115-Mda plasmid. Six of 13 strains tested gave positive cytotoxic and enterotoxic responses. However, two non-cytotoxic strains were enterotoxigenic. Only three of the six cytotoxic and enterotoxic strains caused haemagglutination of human erythrocytes which indicated that toxin production and haemagglutinating activity were unrelated in these *V. cholerae* non-O1 strains. Conjugal transfer assays demonstrated that the 115-Mda plasmid harboured by some of the toxigenic *V. cholerae* non-O1 strains carried genes coding for antibiotic resistance and cytotoxin production but not for enterotoxin production. However, this plasmid was also carried by non-toxigenic strains. Some other strains carrying no plasmids or only small-mol.-wt plasmids, were found to be toxigenic. Therefore, toxin production is not plasmid-mediated in all *V. cholerae* non-O1 strains. Regardless of their pathogenic potential, *V. cholerae* non-O1 strains possessed the capacity to grow in conditions of iron limitation and, under these conditions, synthesis of at least two new outer-membrane proteins was induced.

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10086389 BIOSIS NO.: 199089004280

FUSION OF GENES ENCODING ESCHERICHIA-COLI HEAT-STABLE ENTEROTOXIN AND OUTER MEMBRANE PROTEIN OMP-C

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JOURNAL: Infection and Immunity 57 (11): p3663-3665 1989

ISSN: 0019-9567

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: The OmpC outer membrane protein of *Escherichia coli* was used as a carrier molecule for the nonimmunogenic heat-stable enterotoxin STa. Two fragments of different lengths of the gene encoding STa were fused in vitro to the 3' terminus of the truncated ompC gene. The resulting OmpC-STa hybrid proteins could be detected by L-[35S]cysteine labeling, and they were processed and thus exported. All synthesized hybrid protein remained cell bound and was found by fractionation mainly in the periplasm. Immunoblot analysis showed that the hybrid proteins reacted in vitro both with anti-OmpC and anti-STa antibodies, and immunization of rabbits evoked an antibody response to either of these proteins.

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06945178 BIOSIS NO.: 198376036613

ASSEMBLY IN-VIVO OF ENTERO TOXIN FROM ESCHERICHIA-COLI FORMATION OF THE B SUBUNIT OLIGOMER

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JOURNAL: Journal of Bacteriology 153 (1): p21-26 1983

ISSN: 0021-9193

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: An oligomer of the B subunit of heat-labile *enterotoxin* of *E. coli* was observed in minicells and in whole cells. There is a delay after synthesis of the B subunit before it appears in the oligomer. The delay is not due to slow processing of the precursor. A similar delay in oligomerization of the major *outer* *membrane* *protein* *OmpF* is also described.

1/7/28

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06349435 BIOSIS NO.: 198172083386

ENERGY IS REQUIRED FOR MATURATION OF EXPORTED PROTEINS IN ESCHERICHIA-COLI

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JOURNAL: European Journal of Biochemistry 116 (2): p227-234 1981

ISSN: 0014-2956

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: In numerous cases, proteins which are exported from *E. coli* are synthesized on membrane-bound polysomes in precursor forms which are proteolytically cleaved to generate the mature species. At least 1 step in the export of proteins requires energy. Energy requirements for processing of the precursors of phage M13 coat protein (Date et al., 1980; Date, T., Goodman, L.M., and Wickner, 1980) and the B subunit of heat-labile *enterotoxin* (Palva et al., 1981) were demonstrated previously. An energy requirement for the proteolytic processing of an additional 5 exported proteins (*OmpA* protein, *OmpF* protein, *LamB* protein and 2 periplasmic binding proteins) is reported here. Studies using an *uncA* mutant suggest that the form of energy required is proton motive force. Thus an energized membrane is probably essential for export of most periplasmic and outer membrane proteins.

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05918834 BIOSIS NO.: 198069032821

CHARACTERIZATION OF MEMBRANE BOUND NAD GLYCO HYDROLASE OF VIBRIO-CHOLERAEE
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 JOURNAL: Journal of Biological Chemistry 254 (18): p9254-9261 1979
 ISSN: 0021-9258
 DOCUMENT TYPE: Article
 RECORD TYPE: Abstract
 LANGUAGE: ENGLISH

ABSTRACT: During secretion, enterotoxin is transported across the inner and outer cell membranes of V. cholerae. While attempting to characterize the nature of the toxin fragments while they are being exported out of the cell, proteins which had NADase in isolated inner and outer membrane protein preparations were identified. A MW .apprx. 29,000 protein isolated from the inner membrane demonstrated NADase activity. Most of the NADase activity in the outer membrane was in a 22,000 dalton protein. It is possible that NADase is found as a 29,000 dalton protein in the inner membrane which is processed to form a 22,000 dalton protein in the outer membrane. As the secreted NADase which is associated with the A peptide of cholera toxin has a MW of 21,000, a short peptide probably anchors the 22,000 dalton enzyme to the outer membrane. The inner and outer membrane-associated NADase activities were unlike that associated with pure toxin in that they did not use L-arginine methyl ester as an acceptor for the ADP-ribose released by the hydrolysis of NAD+. The ADP-ribose released by the outer membrane enzyme was free in the reaction mixture, whereas the ADP-ribose formed by the inner membrane enzyme was transferred to an inner membrane protein which had a relative MW of 44,000. This inner membrane protein inhibited DNase and resembled the actin-like membrane-bound elongation factor in Escherichia coli. Antibody prepared against the A fragment of pure toxin inhibited the ADP-ribosyltransferase activities of pure toxin and inner membrane preparations but did not significantly affect the NADase activity. The inner and outer membrane NADase activity and inner membrane ADP-ribosyltransferase activity which may be associated with the membrane-bound precursor of pure toxin are possibly involved in cellular regulation.

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Set	Items	Description
S1	29	ENTEROTOXIN AND (OUTER()MEMBRANE()PROTEIN)

? log y

20jul09 08:57:49	User217744	Session D1221.3
\$6.70	1.083	DialUnits File5
\$70.76	29	Type(s) in Format 7
\$70.76	29	Types
\$77.46		Estimated cost File5
\$0.53		TELNET
\$77.99		Estimated cost this search
\$78.01		Estimated total session cost 1.473 DialUnits

Logoff: level 05.25.00 D 08:57:50